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=> d his

     (FILE 'HOME' ENTERED AT 09:47:15 ON 20 APR 2005)

     FILE 'CAPLUS' ENTERED AT 09:47:28 ON 20 APR 2005
L1             1 S US6368816/PN
               SELECT L1 1 RN
L2        304891 S E1-E11

     FILE 'REGISTRY' ENTERED AT 09:48:02 ON 20 APR 2005
L3             1 S 39433-97-1/RN
               SET NOTICE 1 DISPLAY
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     FILE 'REGISTRY' ENTERED AT 09:48:37 ON 20 APR 2005
L4             1 S 50-22-6/RN
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     FILE 'REGISTRY' ENTERED AT 09:51:22 ON 20 APR 2005
L5             1 S 5697-56-3/RN
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     FILE 'REGISTRY' ENTERED AT 09:51:59 ON 20 APR 2005
L6             1 S 50-99-7/RN
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               SET NOTICE 1 DISPLAY
               SET NOTICE LOGIN DISPLAY

     FILE 'REGISTRY' ENTERED AT 09:52:50 ON 20 APR 2005
L7             1 S 50-23-7/RN
               SET NOTICE 1 DISPLAY
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     FILE 'REGISTRY' ENTERED AT 09:57:47 ON 20 APR 2005
L8             1 S 72-23-1/RN
               SET NOTICE 1 DISPLAY
               SET NOTICE LOGIN DISPLAY

     FILE 'REGISTRY' ENTERED AT 09:59:50 ON 20 APR 2005
L9             1 S 9001-39-2/RN
               SET NOTICE 1 DISPLAY
               SET NOTICE LOGIN DISPLAY
L10            5 S E1-E3,E6-E7

     FILE 'CAPLUS' ENTERED AT 10:04:32 ON 20 APR 2005
L11        50962 S L10
L12         3094 S L11(L)(INSULIN OR DIABET? OR GLUCOS?)
L13         2217 S L11(L)(INSULIN OR DIABET?)
L14         1637 S L13 NOT PY>=1995

     FILE 'USPATFULL, USPAT2' ENTERED AT 10:08:50 ON 20 APR 2005
L15            0 S L14

     FILE 'CAPLUS' ENTERED AT 10:09:09 ON 20 APR 2005

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=> s e1-e11
            85 39433-97-1/BI
         18104 50-22-6/BI
         35878 50-23-7/BI
        176294 50-99-7/BI
         10151 53-06-5/BI
           330 5697-56-3/BI
           690 72-23-1/BI
          5030 9001-39-2/BI
        102426 9004-10-8/BI
          2326 9013-08-5/BI
          1079 9041-46-7/BI
L2      304891 (39433-97-1/BI OR 50-22-6/BI OR 50-23-7/BI OR 50-99-7/BI OR
               53-06-5/BI OR 5697-56-3/BI OR 72-23-1/BI OR 9001-39-2/BI OR
               9004-10-8/BI OR 9013-08-5/BI OR 9041-46-7/BI)
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L7 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2005 ACS on STN
RN **50-23-7** REGISTRY
CN Pregn-4-ene-3,20-dione, 11,17,21-trihydroxy-, (11β)- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Cortisol (8CI)
OTHER NAMES:
CN 11β,17,21-Trihydroxypregn-4-ene-3,20-dione
CN 11β,17,21-Trihydroxyprogesterone
CN 11β,17α,21-Trihydroxypregn-4-ene-3,20-dione
CN 11β-Hydroxycortisone
CN 17-Hydroxycorticosterone
CN 17α-Hydroxycorticosterone
CN 28: PN: US20030109453 SEQID: 27 claimed sequence
CN 4-Pregnene-11β,17α,21-triol-3,20-dione
CN Acticort
CN Aeroseb HC
CN Ala-Cort
CN Anflam
CN Anti-inflammatory hormone
CN CaldeCort Spray
CN CCN 90306A
CN Cetacort
CN Cobadex
CN Cort-Dome
CN Cortanal
CN Cortef
CN Cortenema
CN Corticreme
CN Cortifan
CN Cortiment
CN Cortispray
CN Cortonema
CN Cortril
CN Dermacort
CN Dermocortal
CN Dermolate
CN Dihydrocostisone
CN Dioderm
CN Domolene-HC
CN Efcorbin
CN Efcortelan
CN Eldecort
CN Epiderm H
CN Esiderm H
CN Evacort
CN Ficortril
CN Genacort
CN HC
CN Heb-Cort
CN Hidro-Colisona
CN Hycort
CN Hycortol
CN Hycortole
CN Hydracort
CN Hydrasson
ADDITIONAL NAMES NOT AVAILABLE IN THIS FORMAT - Use FCN, FIDE, or ALL for DISPLAY
FS STEREOSEARCH
DR 8056-08-4, 8063-42-1, 80562-38-5
MF C21 H30 O5
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, AQUIRE, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CEN, CHEMCATS, CHEMINFORMRX, CHEMLIST, CIN, CSCHEM, CSNB, DDFU, DETHERM*, DIOGENES, DRUGU, EMBASE, HODOC*, HSDB*, IFICDB, IFIPAT, IFIUDB, IMSCOSEARCH, IMSPATENTS, IPA, MEDLINE, MRCK*, MSDS-OHS,

NAPRALERT, NIOSHTIC, PHAR, PIRA, PROMT, PS, RTECS*, SPECINFO, SYNTHLINE, TOXCENTER, USAN, USPAT2, USPATFULL, VETU
(*File contains numerically searchable property data)
Other Sources: DSL**, EINECS**, TSCA**, WHO
(**Enter CHEMLIST File for up-to-date regulatory information)

DT.CA CAplus document type: Book; Conference; Dissertation; Journal; Patent; Report

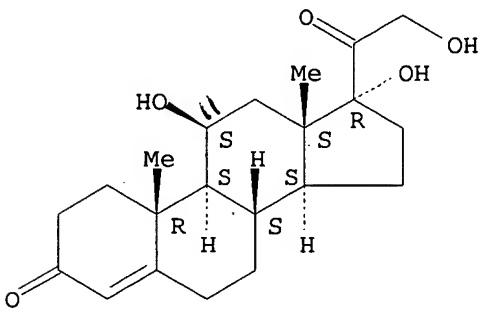
RL.P Roles from patents: ANST (Analytical study); BIOL (Biological study); FORM (Formation, nonpreparative); MSC (Miscellaneous); OCCU (Occurrence); PREP (Preparation); PROC (Process); PRP (Properties); RACT (Reactant or reagent); USES (Uses); NORL (No role in record)

RLD.P Roles for non-specific derivatives from patents: ANST (Analytical study); BIOL (Biological study); PREP (Preparation); PROC (Process); PRP (Properties); RACT (Reactant or reagent); USES (Uses)

RL.NP Roles from non-patents: ANST (Analytical study); BIOL (Biological study); FORM (Formation, nonpreparative); MSC (Miscellaneous); OCCU (Occurrence); PREP (Preparation); PROC (Process); PRP (Properties); RACT (Reactant or reagent); USES (Uses); NORL (No role in record)

RLD.NP Roles for non-specific derivatives from non-patents: ANST (Analytical study); BIOL (Biological study); FORM (Formation, nonpreparative); OCCU (Occurrence); PREP (Preparation); PROC (Process); PRP (Properties); RACT (Reactant or reagent); USES (Uses)

Absolute stereochemistry.

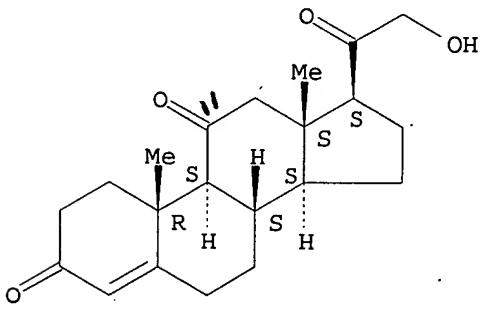


PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

35851 REFERENCES IN FILE CA (1907 TO DATE)
345 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
35878 REFERENCES IN FILE CAPLUS (1907 TO DATE)
20 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L8 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2005 ACS on STN
RN **72-23-1** REGISTRY
CN Pregn-4-ene-3,11,20-trione, 21-hydroxy- (8CI, 9CI) (CA INDEX NAME)
OTHER NAMES:
CN Δ4-Pregnen-21-ol-3,11,20-trione
CN Δ4-Pregnene-21-ol-3,11,20-trione
CN 11-Dehydrocorticosteron
CN 11-Dehydrocorticosterone
CN 11-Oxo-11-deoxycorticosterone
CN 11-Oxocorticosterone
CN 17-(1-keto-2-Hydroxyethyl)-Δ4-androsten-3,11-dione
CN 21-Hydroxypregn-4-ene-3,11,20-trione
CN 4-Pregnen-21-ol-3,11,20-trione
CN Corticosterone, 11-dehydro-
CN Dehydrocorticosterone
CN Kendall's compound A
CN NSC-9702
FS STEREOSEARCH
MF C21 H28 O4
LC STN Files: AGRICOLA, ANABSTR, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CHEMLIST, CSCHEM, DDFU, DRUGU, EMBASE, HODOC*, IFICDB, IFIUDB, MEDLINE, MRCK*, PROMT, TOXCENTER, USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)
DT.CA CAplus document type: Conference; Journal; Patent
RL.P Roles from patents: BIOL (Biological study); FORM (Formation, nonpreparative); PROC (Process); RACT (Reactant or reagent); USES (Uses); NORL (No role in record)
RL.NP Roles from non-patents: ANST (Analytical study); BIOL (Biological study); FORM (Formation, nonpreparative); OCCU (Occurrence); PREP (Preparation); PROC (Process); PRP (Properties); RACT (Reactant or reagent); USES (Uses); NORL (No role in record)
RLD.NP Roles for non-specific derivatives from non-patents: ANST (Analytical study); BIOL (Biological study); OCCU (Occurrence); PRP (Properties); RACT (Reactant or reagent); USES (Uses)

Absolute stereochemistry.

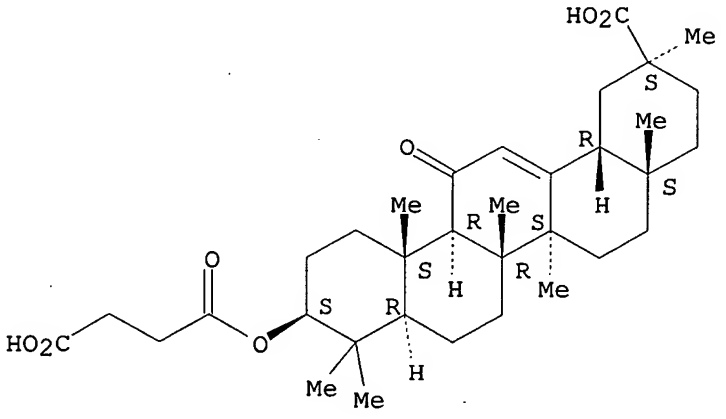


PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

690 REFERENCES IN FILE CA (1907 TO DATE)
8 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
690 REFERENCES IN FILE CAPLUS (1907 TO DATE)
32 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L5 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2005 ACS on STN
RN **5697-56-3** REGISTRY
CN Olean-12-en-29-oic acid, 3-(3-carboxy-1-oxopropoxy)-11-oxo-, (3β,20β)- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Olean-12-en-30-oic acid, 3β-hydroxy-11-oxo-, hydrogen succinate (7CI, 8CI)
CN Olean-12-en-30-oic acid, 3β-hydroxy-11-oxo-, succinate (6CI)
OTHER NAMES:
CN 3-O-(β-Carboxypropionyl)-11-oxo-18β-olean-12-en-30-oic acid
CN 3β-Hydroxy-11-oxoolean-12-en-30-oic acid hydrogen succinate
CN Biogastrone
CN Carbenoxolone
CN Glycyrrhetinic acid hydrogen succinate
FS STEREOSEARCH
DR 13020-80-9, 60093-85-8, 108064-10-4
MF C34 H50 O7
CI COM
LC STN Files: ADISNEWS, AGRICOLA, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CANCERLIT, CAOLD, CAPLUS, CHEMCATS, CHEMLIST, CIN, CSCHEM, DDFU, DRUGU, EMBASE, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, NAPRALERT, PROMT, PS, RTECS*, TOXCENTER, USAN, USPAT2, USPATFULL
(*File contains numerically searchable property data)
Other Sources: EINECS**, NDSL**, TSCA**, WHO
(**Enter CHEMLIST File for up-to-date regulatory information)
DT.CA CAplus document type: Book; Conference; Dissertation; Journal; Patent
RL.P Roles from patents: ANST (Analytical study); BIOL (Biological study); PREP (Preparation); PROC (Process); USES (Uses)
RLD.P Roles for non-specific derivatives from patents: ANST (Analytical study); BIOL (Biological study); USES (Uses)
RL.NP Roles from non-patents: ANST (Analytical study); BIOL (Biological study); MSC (Miscellaneous); PREP (Preparation); PROC (Process); PRP (Properties); USES (Uses); NORL (No role in record)
RLD.NP Roles for non-specific derivatives from non-patents: BIOL (Biological study); PROC (Process); PRP (Properties)

Absolute stereochemistry.



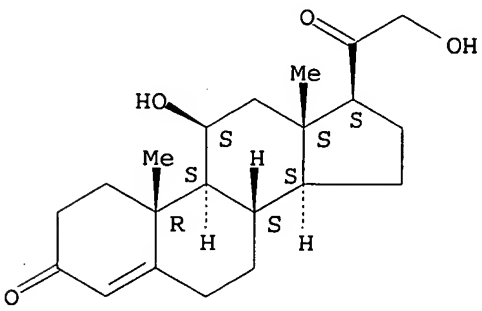
this compound in US 6,368,816

PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

330 REFERENCES IN FILE CA (1907 TO DATE)
5 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
330 REFERENCES IN FILE CAPLUS (1907 TO DATE)
5 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L4 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2005 ACS on STN
RN **50-22-6** REGISTRY
CN Pregn-4-ene-3,20-dione, 11,21-dihydroxy-, (11β)- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Corticosterone (8CI)
OTHER NAMES:
CN 11,21-Dihydroxyprogesterone
CN 11-Hydroxycorticoaldosterone
CN 11β,21-Dihydroxypregn-4-ene-3,20-dione
CN 11β,21-Dihydroxyprogesterone
CN 17-Deoxycortisol
CN 4-Pregnene-11β,21-diol-3,20-dione
CN Corticosteron
CN Kendall's compound B
CN NSC 9705
CN Reichstein's substance H
FS STEREOSEARCH
DR 67085-09-0, 74339-96-1
MF C21 H30 O4
CI COM
LC STN Files: ADISNEWS, AGRICOLA, ANABSTR, AQUIRE, BEILSTEIN*, BIOBUSINESS, BIOSIS, BIOTECHNO, CA, CABA, CANCERLIT, CAOLD, CAPLUS, CASREACT, CBNB, CHEMCATS, CHEMLIST, CIN, CSCHEM, CSNB, DDFU, DRUGU, EMBASE, HODOC*, IFICDB, IFIPAT, IFIUDB, IPA, MEDLINE, MRCK*, NAPRALERT, NIOSHTIC, PROMT, RTECS*, SPECINFO, TOXCENTER, USPAT2, USPATFULL, VETU
(*File contains numerically searchable property data)
Other Sources: EINECS**
(**Enter CHEMLIST File for up-to-date regulatory information)
DT.CA CAplus document type: Book; Conference; Dissertation; Journal; Patent; Report
RL.P Roles from patents: ANST (Analytical study); BIOL (Biological study); FORM (Formation, nonpreparative); OCCU (Occurrence); PREP (Preparation); PROC (Process); PRP (Properties); RACT (Reactant or reagent); USES (Uses); NORL (No role in record)
RLD.P Roles for non-specific derivatives from patents: ANST (Analytical study); BIOL (Biological study); PREP (Preparation); USES (Uses)
RL.NP Roles from non-patents: ANST (Analytical study); BIOL (Biological study); FORM (Formation, nonpreparative); MSC (Miscellaneous); OCCU (Occurrence); PREP (Preparation); PROC (Process); PRP (Properties); RACT (Reactant or reagent); USES (Uses); NORL (No role in record)
RLD.NP Roles for non-specific derivatives from non-patents: ANST (Analytical study); BIOL (Biological study); FORM (Formation, nonpreparative); OCCU (Occurrence); PREP (Preparation); PROC (Process); PRP (Properties); USES (Uses)

Absolute stereochemistry.



PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT

18090 REFERENCES IN FILE CA (1907 TO DATE)
90 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
18104 REFERENCES IN FILE CAPLUS (1907 TO DATE)

2 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L3 ANSWER 1 OF 1 REGISTRY COPYRIGHT 2005 ACS on STN
RN **39433-97-1** REGISTRY
CN Dehydrogenase, 11β-hydroxy steroid (nicotinamide adenine dinucleotide (phosphate)) (9CI) (CA INDEX NAME)
OTHER NAMES:
CN 11β-Hydroxysteroid dehydrogenase
CN 11β-Hydroxysteroid dehydrogenase (NAD(P))
MF Unspecified
CI MAN
LC STN Files: BIOSIS, CA, CAPLUS, TOXCENTER, USPAT2, USPATFULL
DT.CA CAplus document type: Journal; Patent
RL.P Roles from patents: BIOL (Biological study); OCCU (Occurrence); PROC (Process); USES (Uses)
RL.NP Roles from non-patents: BIOL (Biological study); FORM (Formation, nonpreparative); OCCU (Occurrence); PREP (Preparation); PROC (Process); PRP (Properties)

*** STRUCTURE DIAGRAM IS NOT AVAILABLE ***
85 REFERENCES IN FILE CA (1907 TO DATE)
85 REFERENCES IN FILE CAPLUS (1907 TO DATE)

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L14 ANSWER 1 OF 1637 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1995:941022 CAPLUS
DOCUMENT NUMBER: 124:1416
TITLE: Effect of combined administration of counterregulatory hormones during insulin-induced hypoglycemia in rats: Lipolysis mediated by a β-adrenergic mechanism contributes to hyperglycemia
AUTHOR(S): Souza, H. M.; Hell, N. S.; Lopes, G.; Bazotte, R. B.
CORPORATE SOURCE: Departamento de Ciencias Fisiologicas, Universidade Estadual de Londrina, Londrina, 86055-900, Brazil
SOURCE: Brazilian Journal of Medical and Biological Research (1994), 27(12), 2883-7
CODEN: BJMRDK; ISSN: 0100-879X
PUBLISHER: Associacao Brasileira de Divulgacao Cientifica
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The synergistic effect of combined injection of glucagon (G), cortisol (C) and phenylephrine + isoproterenol (E) during hypoglycemia in male adult Wistar rats was investigated. For this purpose the authors injected insulin (1 mg/kg) and 30 min later saline (controls), C (20 mg/kg), G (0.02 mg/kg), or E (1 mg/kg), individually or combined (G+C, G+E, C+E and C+G+E). All drugs were injected i.p. and all rats were killed 60 min after insulin injection. The rise in glycemia with C+G+E was greater (Δ = 107 mg/dL) than the sum of the responses to injection of C, G and E individually, or in double combination plus any single hormone injection. This synergistic effect was reproduced by G + C + isoproterenol (Iso) but not by G + C + phenylephrine (Δ = 0 mg/dL). The results also showed a clear relation between hyperglycemia and lipolysis. Thus, lipolysis mediated by a β-adrenergic mechanism played a significant role in promoting hyperglycemia when Iso was combined with G and C.
IT **50-23-7**, Cortisol 59-42-7, Phenylephrine 7683-59-2, Isoproterenol 9007-92-5, Glucagon, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(counterregulatory hormones effect on lipolysis mediated by β-adrenergic mechanism in **insulin**-induced hypoglycemia)

L14 ANSWER 3 OF 1637 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1995:588747 CAPLUS
DOCUMENT NUMBER: 123:1499
TITLE: A new experimental approach to investigate synergistic interaction of counter-regulatory hormones on glucose recovery after insulin-induced hypoglycemia in rats
AUTHOR(S): Medri de Souza, Helenir; Dall Pizzol, Luciane Isabel; Collar, Raquel Izilda; Ferraz, Marisol; Zanoni, Jacqueline; Bazotte, Roberto Barbosa
CORPORATE SOURCE: Univ. Estadual Londrina, Brazil
SOURCE: Arquivos de Biologia e Tecnologia (1994), 37(4), 737-44
CODEN: ABTTAP; ISSN: 0365-0979
PUBLISHER: Instituto de Tecnologia do Parana
DOCUMENT TYPE: Journal
LANGUAGE: Portuguese
AB The present article describes the exptl. approach to study mechanisms of glucose recovery after acute hypoglycemia induced by high doses of insulin in rats. The results showed that the simultaneous administration of cortisol (C) + glucagon (G) + isoproterenol (iso) were more effective to promote blood glucose recovery from hypoglycemia than the sum of the response of both individual infusion and double combination. The perspective of using this new exptl. approach in future investigations will be discussed.
IT **50-23-7**, Cortisol 7683-59-2, Isoproterenol 9004-10-8, Insulin, biological studies 9007-92-5, Glucagon, biological studies
RL: BAC (Biological activity or effector, except adverse); BSU (Biological

study, unclassified); BIOL (Biological study)
(synergism of cortisol, glucagon and isoproterenol in glucose recovery after **insulin**-induced hypoglycemia)

L14 ANSWER 8 OF 1637 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1995:311939 CAPLUS
DOCUMENT NUMBER: 122:72177
TITLE: Pituitary function tests: comparison of ACTH and 11-deoxy-cortisol responses in the metyrapone test and with the insulin hypoglycemia test
AUTHOR(S): Steiner, H.; Bahr, V.; Exner, P.; Oelkers, P. W.
CORPORATE SOURCE: Dep. Int. Med., Univ. Berlin, Germany
SOURCE: Experimental and Clinical Endocrinology (1994), 102(1), 33-8
CODEN: EXCEDS; ISSN: 0232-7384
PUBLISHER: Barth
DOCUMENT TYPE: Journal
LANGUAGE: English
AB To compare the sensitivity of ACTH and 11-deoxy-cortisol (comp. S) responses in the short metyrapone test and the latter with the insulin hypoglycemia test, retrospective evaluation of 115 short metyrapone tests and comparison of 18 pairs of metyrapone and insulin tests were performed. 20 Healthy controls and 95 patients with confirmed pituitary disease were studied. All hormones were measured by sensitive RIAs. In patients with pituitary disease not requiring hydrocortisone substitution, the ACTH response in the metyrapone test was subnormal in 47 cases (<33 pmol/L), the comp. S. response (<200 nmol/L) in 21 cases only. Comparison of the relation between ACTH and comp. S with an ACTH-cortisol dose-response curve obtained in normal subjects shows that subnormal ACTH responses after metyrapone in the range between 13 and 33 pmol/L still generate normal comp. S responses. The results of the metyrapone test correlated significantly with those of the insulin test. Measuring plasma ACTH in the scope of the metyrapone test makes the test more sensitive to detect secondary adrenal insufficiency than with steroid measurements alone. Results of the metyrapone test correlate significantly with the cortisol response to insulin hypoglycemia.
IT **50-23-7**, Cortisol 152-58-9, 11-Deoxy-cortisol 9002-60-2, ACTH, biological studies
RL: ANT (Analyte); BPR (Biological process); BSU (Biological study, unclassified); ANST (Analytical study); BIOL (Biological study); PROC (Process)
(ACTH and 11-deoxycortisol responses in metyrapone test and cortisol response in **insulin** hypoglycemia test in pituitary function anal.)

L14 ANSWER 10 OF 1637 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1995:262711 CAPLUS
DOCUMENT NUMBER: 122:24252
TITLE: Seasonal changes in body mass, insulin, and glucocorticoids of free-living golden-mantled ground squirrels
AUTHOR(S): Boswell, T.; Woods, S. C.; Kenagy, G. J.
CORPORATE SOURCE: Deps. Zoology and Psychology, Univ. Washington, Seattle, WA, 98195, USA
SOURCE: General and Comparative Endocrinology (1994), 96(3), 339-46
CODEN: GCENA5; ISSN: 0016-6480
PUBLISHER: Academic
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The aim of the present study was to determine the relation between seasonal variation in levels of insulin, cortisol, and corticosterone and body mass in a natural population of golden-mantled ground squirrels (Spermophilus saturatus) over the active season, from Apr. to August. The body mass of females is at an active season min. on emergence from hibernation, increases during pregnancy and lactation, and shows a further rise during fattening, reaching a peak in August. Males are heavier than females on emergence from hibernation, lose mass during the mating period, then

gradually increase to a maximum in August. Plasma insulin titers generally reflected these patterns of change in body mass. Levels in males were almost double those of females on emergence, then decreased as body mass was lost during mating. Male values increased in July and peaked in August, coincident with 6th the body mass maximum Females, in contrast, showed very low insulin levels in Apr. on emergence and increased levels during lactation, continuing high into the fattening period. Corticosterone levels were low in both sexes at the beginning of the season and rose throughout most of the active season; female levels exceeded those of males during the lactation phase. Cortisol titers gradually decreased over the first half of the active season in both sexes, but later increased to a seasonal maximum at the end of the season, coincident with the peak in body mass. Insulin may act as an anabolic agent to promote fat deposition, and also as a signal of total body fat content to influence the transition from mass gain at the end of the active season to mass loss during hibernation. The influence of the glucocorticoids is less clear, but cortisol appears to exert a predominantly anabolic influence on seasonal fattening.

IT **50-22-6**, Corticosterone **50-23-7**, Cortisol 9004-10-8, Insulin, biological studies
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(seasonal changes in body mass and **insulin** and glucocorticoid secretion in free-living golden-mantled ground squirrels)

L14 ANSWER 20 OF 1637 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1994:531428 CAPLUS
DOCUMENT NUMBER: 121:131428
TITLE: Glucose, insulin, and open field responses to immobilization in nonobese diabetic (NOD) mice
AUTHOR(S): Amrani, Abdelazia; Chaouloff, Francis; Mormede, Pierre; Dardenne, Mireille; Homo-Delarche, Francoise
CORPORATE SOURCE: Hop. Necker, Paris, 75015, Fr.
SOURCE: Physiology & Behavior (1994), 56(2), 241-6
CODEN: PHBHA4; ISSN: 0031-9384
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Numerous studies have suggested that stress ppts. type I diabetes. Because stress-elicited hyperglycemia may play a role in this effect, the authors measured the influence of acute immobilization (90 min) upon plasma glucose and insulin levels in nonobese diabetic (NOD) mice, a spontaneous model of type I diabetes. To this end, prediabetic 8-wk-old mice of both sexes were compared to age- and sex-matched C57BL/6 control mice. Baseline plasma glucose levels and immobilization-elicited hyperglycemia were both lower in male and female NOD mice compared to their C57BL/6 counterparts. However, the maximal effects of immobilization upon plasma insulin (and corticosterone) levels were not different between NOD and C57BL/6 mice. When subjected to a metabolic stressor, such as 2-deoxyglucose-induced neuroglucopenia, both strains responded with similar increases in plasma glucose levels. This change was associated with hyperinsulinemia, whose amplitude was lower in NOD than in C57BL/6 females. Lastly, administration of the α2-adrenergic agonist, clonidine, elicited a marked increase in plasma glucose levels, whose amplitude was independent of the strain. The results from this study indicate that the two strains differed in their glycemic response to a psychol., but not to a metabolic, stressor. Because NOD mice were found to exhibit increased locomotion when placed for the first time in an open field, it is suggested that behavioral differences contribute to this differential effect of immobilization upon circulating glucose levels in NOD and C57BL/6 mice.

IT **50-22-6**, Corticosterone 50-99-7, Glucose, biological studies 9004-10-8, Insulin, biological studies
RL: BIOL (Biological study)
(immobilization stress effect on, type I **diabetes** onset in relation to)

L14 ANSWER 26 OF 1637 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1994:320604 CAPLUS

DOCUMENT NUMBER: 120:320604
TITLE: Effects of chronic hypercortisolemia on carbohydrate metabolism during insulin deficiency
AUTHOR(S): Goldstein, Richare E.; Cherrington, Alan D.; Reed, George W.; Lacy, D. B.; Wasserman, David H.; Abumrad, Naji N.
CORPORATE SOURCE: Sch. Med., Vanderbilt Univ., Nashville, TN, 37232, USA
SOURCE: American Journal of Physiology (1994), 266(4, Pt. 1), E618-E627
CODEN: AJPHAP; ISSN: 0002-9513
DOCUMENT TYPE: Journal
LANGUAGE: English

AB This study was undertaken to further investigate the effect of acute selective insulin deficiency on glycogenolysis and gluconeogenesis occurring during chronic physiol. hypercortisolemia in conscious overnight fasted dogs. After an 80-min tracer and dye equilibration period and a 40-min basal period, selective insulin deficiency was created during the 180-min exptl. period by infusing somatostatin peripherally (0.8 μg·kg-1·min-1) with basal replacement of glucagon intraportally (0.65 ng·kg-1·min-1). In the cortisol group (n = 5), a continuous infusion of hydrocortisone (3.5 μg-1·min-1) was begun 5 days before the experiment In the saline group (n = 5), there was no infusion of cortisol. [3-3H]glucose, [U-14C]alanine, and indocyanine green dye were used to assess glucose production and gluconeogenesis using tracer and arteriovenous difference techniques. During selective insulin deficiency in the saline group, the arterial plasma glucose level (Glc) increased from 109 ± 2 to 285 ± 19 mg/dL; glucose production increased from 2.7 ± 0.2 to 4.5 ± 0.3 mg·kg-1·min-1. Gluconeogenic efficiency and conversion of alanine to glucose (Conv) increased by 300 ± 55 and 356 ± 67%. During selective insulin deficiency in the cortisol group, Glc increased from 117 ± 3 to 373 ± 50 mg/dL; glucose production increased from 3.3 ± 0.5 to 6.9 ± 0.7 mg·kg-1·min-1. Gluconeogenic efficiency and Conv increased by 268 ± 41 and 393 ± 75%, resp. The maximal glycogenolytic rate increased significantly more in the cortisol group than in the saline group, accounting for the difference in glucose production These results suggest that, even during chronic hypercortisolemia, acute insulin deficiency has more pronounced effects on glycogenolysis than gluconeogenesis.

IT **50-23-7**, Cortisol
RL: BIOL (Biological study)
(metabolic disorders, hypercortisolemia, glycogenolysis and gluconeogenesis in, in **insulin** deficiency)

L14 ANSWER 27 OF 1637 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1994:320587 CAPLUS
DOCUMENT NUMBER: 120:320587
TITLE: Metabolic effects of hypoglycemic counterregulation during sustained mild hyperinsulinemia and constant glucose availability in healthy men
AUTHOR(S): Godfried, Mieke H.; Romijn, Johannes A.; Endert, Erik; Sauerwein, Hans P.
CORPORATE SOURCE: Dep. Intern. Med., Univ. Amsterdam, Amsterdam, Neth.
SOURCE: Nutrition (New York, NY, United States) (1994), 10(1), 5-10
CODEN: NUTRER; ISSN: 0899-9007
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Posthypoglycemic metabolic changes have been studied predominantly during waning of insulin action. The authors evaluated the effects of hypoglycemic counterregulation on glucose and lipid metabolism during continuous insulin infusion. Glucose was infused at a constant rate throughout the study (4 mg · kg-1 · min-1). During the second part of the study, plasma glucose levels were clamped at .apprx.4 mM by variable insulin infusion. In six subjects, but not in five control subjects, short-term hypoglycemia (nadir plasma glucose 2.80 ± 0.05 mM) was induced by an addnl. bolus injection of insulin before starting insulin infusion. Substrate oxidation rates and plasma substrate fluxes were

determined by indirect calorimetry and primed continuous infusions of [3-3H]glucose and [14C]palmitate. After hypoglycemia, higher insulin infusion rates than in the control group were required to clamp plasma glucose concns. at similar levels ($p < 0.05$). Addnl., insulin levels were increased compared with those in control subjects ($p < 0.01$). There were no differences in substrate oxidation rates, hepatic glucose production, or lipolysis after hypoglycemia. In conclusion, the counterregulatory hormonal response after short-lasting mild hypoglycemia with sustained modest hyperinsulinemia and constant glucose availability induces insulin resistance with respect to glucose uptake but is unable to stimulate hepatic glucose production or lipolysis.

IT **50-23-7**, Cortisol 51-41-2, Norepinephrine 51-43-4, Epinephrine 9007-92-5, Glucagon, biological studies
RL: PRP (Properties)
(short-term hypoglycemia induction of, **insulin** resistance in response to, in healthy humans in presence of sustained mild hyperinsulinemia and constant glucose availability)

L14 ANSWER 29 OF 1637 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1994:214297 CAPLUS
DOCUMENT NUMBER: 120:214297
TITLE: The effects of acute hypercortisolemia on β-hydroxybutyrate and glycerol metabolism during insulin deficiency
AUTHOR(S): Goldstein, R. E.; Wasserman, D. H.; Reed, G. W.; Lacy, D. B.; Abumrad, N. N.; Cherrington, A. D.
CORPORATE SOURCE: Sch. Med., Vanderbilt Univ., Nashville, TN, USA
SOURCE: Hormone and Metabolic Research (1994), 26(1), 9-13
CODEN: HMMRA2; ISSN: 0018-5043
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The present study was undertaken to determine whether an acute physiol. rise in plasma cortisol during selective insulin deficiency would have significant effects on glycerol and β-hydroxybutyrate metabolism in conscious overnight-fasted dogs. Each experiment consisted of a two hour dye equilibration period, a 40 min basal period, and a 3 h exptl. period. A continuous infusion of indocyanine green dye for blood flow estimation was initiated at the start of the equilibration period and continued throughout the experiment In both of two protocols selective insulin deficiency was created during the exptl. period by infusing somatostatin peripherally (0.8 μg/kg.min) with basal replacement of glucagon intraportally (0.65 ng/kg-min). In the test protocol (Cortisol, n = 5), 3.0 μg/kg-min of hydrocortisone was infused during the exptl. period. In the control protocol (Saline, n = 5), saline was infused. Et hepatic balances were determined using the (A-V) difference technique. During selective insulin deficiency alone (Saline), the arterial blood glycerol and level increased from 81 ± 19 to 140 ± 11 μM ($p < 0.01$) and net hepatic glycerol uptake (NHGlyU) tended to increase from 2.3 ± 0.3 to 3.3 ± 0.6 μmol/kg-min ($0.05 < 0.1$). The arterial plasma free fatty acid (FFA) level remained unchanged at 1041 ± 35 μM. The arterial β-hydroxybutyrate (BHOB) level increased slightly from 21 ± 4 to 29 ± 5 μM while net hepatic β-hydroxybutyrate production (NHBP) remained unchanged (1.0 ± 0.2 μmol/kg-min). During acute hypercortisolemia with selective insulin deficiency (Cortisol), similar changes occurred in the arterial blood glycerol level and net hepatic glycerol uptake. The arterial plasma FFA level remained unchanged at 1214 ± 105 ± μM. However, the BHOB level increased from 19 ± 3 to 67 ± 10 μM while NHBP increased from 1.1 ± 0.1 to 2.8 ± 0.6 μmol/kg-min. These results suggest that an acute physiol. rise in plasma cortisol during insulin deficiency can markedly enhance ketogenesis and that this enhancement occurs independently of changes in lipolysis.

IT **50-23-7**, Cortisol
RL: BIOL (Biological study)
(metabolic disorders, acute hypercortisolemia, β-hydroxybutyrate and glycerol metabolism response to, in **insulin** deficiency)

L14 ANSWER 31 OF 1637 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1994:209429 CAPLUS

DOCUMENT NUMBER: 120:209429
TITLE: Hormonal regulation and biological actions of insulin-like growth factor binding proteins in isolated ovine thyroid follicles
AUTHOR(S): Phillips, Ian D.; Becks, Gregory P.; Wang, Jia Fang; Han, Victor K. M.; Hill, David J.
CORPORATE SOURCE: Lawson Res. Inst., St. Joseph's Health Cent., London, ON, N6A 4V2, Can.
SOURCE: Endocrinology (1994), 134(3), 1238-46
CODEN: ENDOAO; ISSN: 0013-7227
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The purposes of these studies was to 1) further characterize the species of IGFBPs synthesized by thyroid follicles, 2) examine the ability of TSH and cortisol to alter IGFBP gene expression and protein release, and 3) investigate the actions of exogenous IGFBPs on thyroid cell function. Adult sheep thyroid follicles were isolated after collagenase digestion, grown to confluence in Coon's modified Ham's F12M medium (0H) with the addition of transferrin, glycyl-histidyl-lysine, somatostatin (3H), cortisol and insulin, and maintained in serum-free test media with or without further supplements for ≤48 h. Conditioned media were analyzed for IGFBP presence by Western ligand blotting, and by immunoblotting using specific antisera against bovine IGFBP-2 and human IGFBP-5. IGFBP mRNAs from follicles were identified by Northern blot hybridization using [32P]labeled complementary DNAs encoding ovine IGFBP-1 or -2, and rat IGFBP-4, -5, or -6. Uptake and organification of Na[125I] were measured by incorporation into trichloroacetic acid-precipitable material. Isolated thyroid follicles synthesized four species of IGFBPs in either 0H or 3H medium as detected by ligand blotting, of sizes 40-46, 34, 28, and 18 kDa (kDa), resp. The 32 kDa IGFBP was identified immunol. as IGFBP-2, whereas the 28 kDa and 18 kDa species were identified as IGFBP-5. Northern blot hybridization of total RNA from cells in 3H medium demonstrated an IGFBP-2 mRNA [1.4 kilobase (kb)], an IGFBP-4 mRNA (2.6 kb), and two IGFBP-5 mRNAs (6 kb and 1.8 kb). No IGFBP-1 or -6 mRNAs were detected. Incubation of cultured follicles with TSH (30-500 μU/mL) caused dose-dependent decrease in the abundance of all IGFBP mRNAs and released proteins, which were reduced further by TSH together with cortisol (10 nM). When the inhibitory effect of TSH and cortisol was removed, IGFBP-2 mRNA increased within 3 h and was 7-fold greater within 12 h. IGFBP-2 did not appear in the conditioned medium until 12 h after TSH removal, along with the other IGFBP species. Incubation of follicles with TSH, in either 0H or 3H medium, with or without the further addition of cortisol alone, or in combination with either IGF-I (6.7 nM) or insulin (1.67 μM), caused an increase in iodine uptake and incorporation which was blocked in the presence of exogenous bovine IGFBP-2, but not by human IGFBP-3. The result show that ovine thyroid follicles synthesized two major species of IGFBPs, IGFBPs -2 and -5, which are regulated acutely by TSH. The rapid changes in local release of IGFBP-2 may modulate the actions of exogenous or endogenous IGFs, and may be one important mechanism whereby TSH regulates iodide transport and its organification in thyroid follicles.

IT **50-23-7**, Cortisol
RL: BIOL (Biological study)
(**insulin**-like growth factor binding proteins formation and secretion response to TSH and, in thyroid follicles)

L14 ANSWER 36 OF 1637 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1994:46225 CAPLUS
DOCUMENT NUMBER: 120:46225
TITLE: Expression of 11β-hydroxysteroid dehydrogenase mRNA in rat vascular smooth muscle cells
AUTHOR(S): Takeda, Y.; Miyamori, I.; Yoneda, T.; Ito, Y.; Takeda, R.
CORPORATE SOURCE: Sch. Med., Kanazawa Univ., Kanazawa, 920, Japan
SOURCE: Life Sciences (1994), 54(4), 281-5
CODEN: LIFSAK; ISSN: 0024-3205
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The enzyme 11β-hydroxysteroid dehydrogenase (11-HSD) converts corticosterone to the inactive 11-dehydrocorticosterone in the rat. The bioactivity of 11-HSD, expressed as the percentage conversion of 3H-corticosterone to 3H-11-dehydrocorticosterone, was 13.7% in rat vascular smooth muscle cells (rVSMC). Cells treated with 100 nM dexamethasone (Dex) showed a 1.4-fold increase in 11-HSD activity. Insulin (100 μM) decreased 11-HSD activity by 0.8-fold. Expression of 11-HSD mRNA was also confirmed in rVSMC by Northern blot anal. Dexamethasone increased and insulin decreased the levels of 11-HSD mRNA in parallel with the increase in bioactivity. Vascular smooth muscle cells express 11-HSD activity; the access of corticosterone to vascular smooth muscle receptors may be modulated by the enzyme.

IT **72-23-1**, 11-Dehydrocorticosterone
RL: BIOL (Biological study)
(corticosterone conversion to, in blood vessel smooth muscle cells, **insulin** and glucocorticoid effect on)

IT **50-22-6**, Corticosterone
RL: BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); PROC (Process)
(metabolism of, to dehydrocorticosterone, in blood vessel smooth muscle cells, **insulin** and glucocorticoid effect on)

L14 ANSWER 52 OF 1637 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1993:121347 CAPLUS
DOCUMENT NUMBER: 118:121347
TITLE: Corticosterone effect on insulin receptor number and kinase activity in chicken muscle and liver
AUTHOR(S): Taouis, Mohammed; Derouet, Michel; Chevalier, Bernadette; Simon, Jean
CORPORATE SOURCE: Stn. Rech. Avic., INRA, Nouzilly, 37380, Fr.
SOURCE: General and Comparative Endocrinology (1993), 89(2), 167-75
CODEN: GCENA5; ISSN: 0016-6480
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The effects of chronic corticosterone treatment (6 mg/kg/day) on insulin sensitivity and on liver and muscle insulin receptors were examined in 5-wk-old chickens. The hypoglycemic effect of exogenous insulin was completely abolished within 2 wk of treatment, suggesting a corticosterone-induced insulin resistance. Hepatic insulin receptor nos. were slightly reduced after 2 wk of treatment. After 1 or 2 wk, corticosterone treatment reduced liver insulin receptor kinase activity toward the artificial substrate poly(Glu4,Tyr1). Muscle insulin receptor kinase activity was also decreased after 1 wk of treatment, but this effect was accounted for by a decrease in basal activity. Therefore, the corticosterone-induced insulin resistance is accounted for, at least in part, by altered hepatic receptor nos. and kinase activity. The impairment of muscle development involves postreceptor defects.

IT **50-22-6**, Corticosterone
RL: BAC (Biological activity or effector, except adverse); BSU (Biological study, unclassified); BIOL (Biological study)
(**insulin** receptor number and tyrosine kinase activity response to, in liver and muscle)

L14 ANSWER 54 OF 1637 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1993:16631 CAPLUS
DOCUMENT NUMBER: 118:16631
TITLE: Effects of acute and chronic counterregulatory hormone infusions on glucose tolerance and insulin sensitivity in diabetic dogs
AUTHOR(S): Sleeman, Mark W.; Christopher, Michael J.; Martin, Iva K.; Ward, Glenn M.; Alford, Frank P.; Best, James D.
CORPORATE SOURCE: Dep. Med., Univ. Melbourne, Fitzroy, Australia
SOURCE: Diabetes (1992), 41(11), 1446-32
CODEN: DIAEAZ; ISSN: 0012-1797
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The effects of elevated epinephrine (EPI) and cortisol (CORT) levels on

glucose tolerance (KG), insulin sensitivity (SI), and glucose-mediated glucose disposal (SG) were studied in dogs with alloxan-induced diabetes. Conscious dogs received saline (SAL), EPI at 20 ng/kg/min for 30 min (short EPI) or 72 h (long EPI), or CORT at 200 μg/kg/min for 60 min (short CORT) or 72 h (long CORT) before assessment of glucose metabolism by rapid sampling for glucose and insulin levels after 300 mg/kg i.v. glucose and exogenous insulin infusion designed to simulate the normal secretory pattern. With EPI infusion, KG fell acutely from 2.9 to 2.0%/min (SAL vs. short EPI), but rose to 3.4%/min during long EPI. Minimal-model anal. of the glucose response with the insulin data as input showed that SI decreased acutely from 4.7 to 2.5 + 10-5/min/pM (SAL vs. short EPI), but rose to 4.5 + 10-5/min/pM during long EPI. The effects of EPI on SG paralleled the results for KG and SI, with acute decline from 3.9 to 2.1 + 10-2/min (SAL vs. short EPI) and recovery to 3.3 + 10-2/min during long EPI. During CORT infusion, KG tended to fall (SAL 2.9 vs. short CORT 2.5 vs. long CORT 2.2%/min). This decline was related to a fall of SI (SAL 4.7 vs. short CORT 2.7 vs. long CORT 1.2 + 10-5/min/pM, long CORT vs. SAL), whereas SG levels were similar for the 3 groups. These results indicate that, in the absence of any compensatory change of insulin secretion, adaptation to the metabolic effects of long-term hormone elevation occurs for EPI but not CORT, which has a sustained effect on SI. Therefore, CORT may be more important than EPI as a contributor to long-term stress-induced hyperglycemia in people with type I diabetes.

IT **50-23-7**, Cortisol 51-43-4, Epinephrine
RL: BIOL (Biological study)
(glucose tolerance and **insulin** sensitivity response to acute and chronic administration of, in **diabetes**)

L14 ANSWER 61 OF 1637 CAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1992:626655 CAPLUS
DOCUMENT NUMBER: 117:226655
TITLE: Influence of growth hormone and cortisol on insulin actions in the growing ruminant
AUTHOR(S): Jackiw, Maria
CORPORATE SOURCE: Univ. Newcastle upon Tyne, Newcastle upon Tyne, UK
SOURCE: (1990) 309 pp. Avail.: Univ. Microfilms Int., Order No. BRDX93804
From: Diss. Abstr. Int. B 1992, 52(7), 3356-7
DOCUMENT TYPE: Dissertation
LANGUAGE: English
AB Unavailable
IT **50-23-7**, Cortisol 9002-72-6, Growth hormone
RL: BIOL (Biological study)
(**insulin** action response to, in development in ruminant)